Archive of Obstetrics, Gynecology and Reproductive Medicine
AOGRM, 2(1): 38-39
www.scitcentral.com
ISSN: 2640-2297

Commentary: Open Access

Treatment of Diabetic Ketoacidosis and Coma in Pregnant Women

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Received January 07, 2019; Accepted January 30, 2019; Published January 31, 2019

Several papers have discussed the problems in the treatment of diabetic ketoacidosis and coma in pregnant women [1-3]. Now, it is the time to summarize the results of these discussions.

Substantial progress has been made possible with the Nobel-prize 1977 to Yallow for the development of new methods of biochemical analysis that it make possible to measure insulin concentration in human plasma. Since the general acceptance of the hyperosmolar hyperglycemic non-ketonic coma [4] has emerged a complicated situation: how is it possible to explain with insulin deficiency two diametric different phenomena, diabetic ketoacidotic coma and hyperosmolar hyperglycemic non-ketotic coma? Usually, a "very simple" explanation has been proposed; absolute deficiency of plasmatic insulin is the cause of diabetic ketoacidotic coma, relative deficiency of insulin is the cause of hyperosmolar hyperglycemic non-ketotic coma. However, the application of Yallow's methods to measure insulin concentration has shown that this is not correct. 1981 has been published the monograph "Diabetic coma: ketoacidotic and hyperosmolar" [5] and on page 67, Figure 6.3 has the names of 12 authors who have reported sufficient amounts of plasmatic insulin in patients with diabetic ketoacidosis. In contrast, absolute deficiency of plasmatic insulin has been reported in diabetic patients with hyperosmolar hyperglycemic nonketotic coma [6]. On the opinion that absolute deficiency of plasmatic insulin is the cause of diabetic ketoacidosis and coma has been casted doubts already 1973. Munro et al. [7] have reported patients with euglycemic diabetic ketoacidosis and, thus, the question aroused whether insulin deficiency is possible in euglycemia?

The solution of this problem has been made by several publications [8] reporting that the immediate cause of decreased level of consciousness up to coma is decreasing blood-pH (=increasing concentration of hydrogen ions H+). The glycolytic enzyme phosphofructokinase is pH-dependent, as its activity is decreasing with decreasing pH and, thus, is decreased also the utilisation of glucose in brain cells (regardless what is the concentration of glucose in the blood). The decrease of blood-pH is caused by increased concentration of up to 36 organic acids [9]; including also reports on very low blood-pH without increase of acetoacetic and beta-hydroxybutyric acids [10].

In pregnant women, most dangerous is the situation, if the patient is admitted in coma. The treatment with sodium bicarbonate should begin as soon as possible to avoid irreversible damage of the brain cells caused by low blood-pH. If a diabetic pregnant woman is on insulin therapy, this should be carefully administered to avoid the development of hyperosmolar hyperglycemic non-ketotic coma. The blood-pH should be checked in regular intervals and if this is decreased, therapy with sodium bicarbonate infusion is indicated until normal values have been reached and a continuation of the therapy in the hospital should be considered.

SUMMARY

The methods to measure insulin concentration in the blood has made possible better understand the treatment of diabetic ketoacidotic coma and hyperosmolar hyperglycemic non-ketotic coma also in pregnant diabetic women. Successful treatment of ketoacidotic coma is infusions of sodium bicarbonate, insulin administration is important for prevention and treatment of hyperosmolar hyperglycemic non-ketotic coma.

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Citation: Rosival V. (2019) Treatment of Diabetic Ketoacidosis and Coma in Pregnant Women. Arch Obstet Gynecol Reprod Med, 2(1): 38-39.

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